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2F1 antigen, the mouse homolog of the rat "mast cell function-associated antigen", is a lectin-like type II transmembrane receptor expressed by natural killer cells.

Hanke T, Corral L, Vance RE, Raulet DH.

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Department of Molecular and Cell Biology, University of California, Berkeley
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Inhibitory lectin-like receptors expressed on the surface of hematopoietic cells are critically involved in regulation of their effector functions. Here we report that a novel mAb specific for mouse NK cells, 2F1, recognizes the mouse homolog of the mast cell function-associated antigen (MAFA), an inhibitory lectin-like transmembrane receptor expressed on rat mast cells. The 2F1 antigen (2F1-Ag) and rat MAFA are structurally highly conserved and contain a cytoplasmic motif similar to the immunoreceptor tyrosine-based inhibitory motif that is presumably utilized for inhibitory signaling. We also identified a human homolog that is closely related to the rodent MAFA/2F1-Ag proteins. Like rat MAFA, 2F1-Ag is probably encoded by a single gene, which exhibits relatively little polymorphism. Strikingly, while rat MAFA is considered a mast cell antigen, we have been unable to detect cell surface expression of 2F1-Ag by mouse mast cell lines, bone marrow-derived mast cells, or peritoneal mast cells. Furthermore, mouse bone marrow-derived mast cells were devoid of 2F1-Ag mRNA. Instead, we find that approximately 40% of mouse NK cells express 2F1-Ag. Thus, MAFA/2F1-Ag may modulate immunological responses on at least two different cell types bridging the specific and innate immune system.

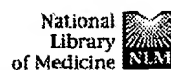
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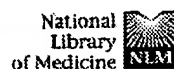
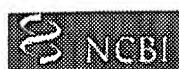
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T lymphocyte-mediated antiviral immune responses in mice are diminished by treatment with monoclonal antibody directed against the interleukin-2 receptor.

Utermohlen O, Tarnok A, Bonig L, Lehmann-Grube F.

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Heinrich-Pette-Institut für Experimentelle Virologie und Immunologie an der Universität Hamburg, Germany.

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Blocking the interleukin-2 receptor's alpha-chain in lymphocytic choriomeningitis virus-infected mice by treatment with monoclonal antibodies diminished the increase of numbers of CD8+ T lymphocytes in spleens and prevented CD8+ T lymphocyte-mediated virus clearance from organs as well as generation of virus-specific cytotoxic T lymphocytes. Also, the CD8+ T cell-mediated early phase of the delayed-type hypersensitivity footpad swelling reaction was decreased. The same treatment had no effect on the number of CD4+ spleen T lymphocytes, which, however, did not enlarge during infection, but these cells' heightened DNA synthesis and cytokine production were reduced by antibody treatment; yet the generation of antiviral antibodies remained unaffected, and the CD4+ T lymphocyte-mediated second part of the footpad reaction was somewhat augmented. We conclude that blocking of the interleukin-2 receptor by antibody in lymphocytic choriomeningitis virus-infected mice diminishes both CD8+ and CD4+ T cell-mediated antiviral immune responses, the former more than the latter.

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